cancer in particular (Liddell 1973b). There was a wide disparity between the SMR of miners employed at the face (49) and the SMR of those working on the surface (82). This disparity can be explained in that surface miners may smoke while at work, but underground miners cannot, and that the less healthy miners tend to move away from coal face work and to be employed in easier jobs on the surface.

Ortmeyer and colleagues (1973; Lainhart et al. 1969) studied the death rate of Pennsylvania coal miners who had been awarded compensation for occupationally related respiratory disability. The overall SMR was the same as that of white men in Pennsylvania. Excess death rates were found in subjects with a reduced ventilatory capacity, in particular when the FEV_1/FVC was below 50 percent, and in ex-miners with stage B and C complicated CWP. "Disabled" miners with simple CWP had a normal SMR. Later, Ortmeyer and colleagues (1974) studied a group of randomly selected Appalachian miners and ex-miners. They showed that the overall life expectancy for miners and ex-miners combined was the same as that for the general U.S. population and for the States from which the cohort originated. The effects on mortality of the years worked underground, cigarette smoking, and airways obstruction were investigated. Ex-miners had a slightly but significantly increased death rate. Simple CWP had no effect on life expectancy; however, complicated CWP was associated with decreased longevity. Although both cigarette smoking and airways obstruction were associated with an increased SMR, the number of years of work underground had no discernible effect.

About the same time these reports were published, Cochrane (1973) described his findings from a 20-year followup of the male population of the Rhondda Fach in Wales. Survival rates for miners and ex-miners were independent of the radiographic presence of simple CWP or category A complicated pneumoconiosis. Further studies of the same cohort from the Rhondda Fach showed a normal life expectancy in those with simple CWP and category A complicated CWP (Cochrane et al. 1979). The death rate for bronchitis and other respiratory diseases was elevated. The smoking habits of this population were not examined.

A 20-year followup of a population sample from Derbyshire (Cochrane and Moore 1980), aged 25 to 34, included four groups of workers whose work was categorized as nondusty, pure coal mining, pure foundry, or other and mixed. There were no significant differences in the SMRs of the dust-exposed group of miners, compared with the non-dust-exposed group of miners; however, only 20 deaths were recorded in the study.

Rockette (1977), in a NIOSH-supported study, investigated the mortality rates among coal miners covered by the United Mine Workers of America (UMW) Health and Retirement Fund. Unfortu-

nately, unlike the studies of Ortmeyer and colleagues (1973, 1974), in this investigation the population was not randomly chosen and smoking histories were not available. A 10 percent sample of miners eligible for benefits in January 1959 was randomly selected from the original 550,000. Doubt exists whether miners covered by the UMW Health and Retirement Fund are necessarily representative of U.S. coal miners as a whole. The SMR for all causes was 101.6 and for all cancer, 97.7. The SMRs for asthma, emphysema, and tuberculosis were significantly elevated at 174, 144, and 145, respectively. The SMR for lung cancer was minimally but significantly elevated at 112; however, a disproportionate number of miners from southwestern West Virginia were subsequently shown to have been included in the cohort. The death rate for lung cancer in this part of the United States is significantly higher than the rate for the United States as a whole and for other parts of West Virginia. In the absence of smoking histories, little can be made of such a small increment in mortality. Deaths due to accidents were high (SMR 144). The SMR for bronchitis was 89.7, but, as previously mentioned, the SMR for emphysema was elevated. There was a significantly increased death rate for stomach cancer, but the SMR for pneumoconiosis could not be determined because of difficulties with death certification and classification.

A lengthy report of coal miners' morbidity in relation to x-ray category, lung function, and exposure to airborne dust (Miller et al. 1981) described the findings for 31,611 British miners who were surveyed at 24 pits from 1953 to 1958. Again, the coal miners had a lower mortality than British men in general. Although this was attributed to the healthy worker effect, no supporting evidence for this conclusion was given. Miners with PMF had an increased death rate. Mortality from bronchitis was associated with increased dust exposure, but it is apparent that with increased dust exposure, there would also be an increased cumulative exposure to cigarette smoke. Appropriate data whereby the two effects could be separated were not available, since cumulative cigarette smoking history or, indeed, a smoking history of any kind was not available. Deaths from lung cancer were not increased.

Higgins and colleagues (1981), in a followup study of a group of miners from Richwood and Mullens, West Virginia, were unable to show any significant difference in the mortality of miners and ex-miners as compared with nonminers. The death rates from respiratory diseases were appreciably higher in coal miners; however, there are doubts as to the accuracy of the cause of death on the death certificates because compensation was often awarded to ex-miners' families solely on the basis of a death certificate that mentioned respiratory disease (Comptroller General 1980, 1982). Another problem encountered was that some miners had moved away from

the district and could not be traced. In many instances, their status proved impossible to determine. A clear-cut effect of smoking on mortality was evident in nonminers, but was less evident in miners and ex-miners. Here again the advent of black lung compensation may have been an incentive for disability applicants to underestimate their smoking habits.

The publication of the Registrar General's Decennial Supplement (1970–1972) on occupational mortality (1978) indicated that mortality rates for coal miners were somewhat increased for both underground and surface workers. The SMR for most respiratory diseases other than lung cancer showed a mild to moderate increase.

Jacobsen (1976, 1977) concluded that coal miners as a group have a normal SMR. He also indicated that there was no excess death rate from bronchitis and emphysema among coal miners, nor was there an increase in mortality from these conditions with increasing time worked in dusty occupations. Among men with no pneumoconiosis, there was a clear and significant mortality gradient with increases in estimates of cumulative exposures to airborne dust. However, the decision to study the SMR of selected subgroups of miners whose cigarette smoking habits were unknown, and in whom other possible confounding factors may have been present, detracts from his conclusions. The demonstration that the presence of bronchitis in coal miners is associated with increased mortality and morbidity is of little special significance for coal miners because the same situation applies to the general population. The increased mortality and morbidity are for the most part attributable to cigarette smoking in the general population, and only if it were possible to show an increased death rate in nonsmoking bronchitic coal miners would this observation be convincing evidence that the presence of bronchitis of itself portends premature disability and death.

In conclusion, the majority of recent mortality studies have shown that coal miners have a normal life expectancy. Although there is an increased SMR in miners with PMF, the overall prevalence of PMF in working miners is so low that any effect it has on the SMR is more than counterbalanced by decreased death rates from lung cancer and heart disease. Although in certain studies, death rates from bronchitis and emphysema have been found to be elevated, this has not been a consistent finding; in other studies, especially those in which it has been possible to quantitate the effects of cigarette smoking, no increased death rates have been demonstrable. There is little or no evidence that the inhalation of coal mine dust contributes to excess morbidity or mortality in regard to lung conditions other than PMF, such as emphysema, asthma, tuberculosis, or pneumonia. By way of contrast, cigarette smoking has repeatedly been shown to have a clear and easily demonstrable effect on the death rate of both miners and nonminers. There is some recent suggestion that cigarette

smoking prevalence increased in British coal miners between 1965 and 1975, possibly related to their increased standard of living, but it is too early for any discernible changes in cigarette consumption to be reflected in mortality and morbidity statistics.

Lung Function in Coal Miners

Although there is no substantial clinical effect of an increasing category of simple CWP on the ventilatory capacity of coal miners, most studies in which coal miners have been compared with a suitable reference population of nonminers have demonstrated a significant decrement in the ventilatory capacity of the miners (Higgins 1972). Regardless of radiographic evidence of simple CWP, FEV_1 of coal miners—or any other suitable index of ventilatory capacity—is generally reduced in comparison with FEV_1 of nonminers. This suggests that decrements in FEV_1 and simple CWP are both related to dust exposure, but the two measures represent separate biologic responses in the lung to the inhalation of coal dust.

Higgins studied three populations in the United Kingdom, all of which contained a significant proportion of miners and ex-miners, along with a comparable reference population. The areas chosen were Leigh, the Rhondda Fach, and Stavely (Higgins 1960; Cochrane et al. 1961). The reduction in the ventilatory capacity of miners that was observed could not be explained on the basis of cigarette smoking; indeed, coal miners at that time generally smoked less than nonminers. Since then, several other studies have found similar results and the data have been reviewed by Higgins (1972). Possible explanations for the observation that coal miners have a lower ventilatory capacity and that this finding is unrelated to radiographic findings are that (1) coal dust can produce or exacerbate emphysema or airway narrowing and that these changes occur independent of the changes that result in an abnormal radiograph, or (2) the lower ventilatory capacity in miners results from either industrial selection or differential migration. Thus, were the more healthy miners to leave their employment and move to other parts of the country to seek new jobs, those who remain would be less healthy and almost certainly have lower lung function. Although the second hypothesis is a consideration, especially during hard times when unemployment in the coal mines is high, recent studies have shown that it is the weaker and the less muscular man who is more likely to leave the coal mine within the first few months of his employment (McLintock 1971). Thus, the first hypothesis seems much more probable and requires further consideration.

Emphysema, Exposure to Coal Dust, and Cigarette Smoking

The pathology associated with CWP, both simple and complicated, has been well described, and it is generally accepted that simple CWP has a relatively specific set of histological findings (College of American Pathologists 1979). Initially, dust starts to accumulate around the second division of respiratory bronchioles. As this occurs, there may be a little reticulin or, exceptionally, some collagenous fibrosis. Subsequently, the respiratory bronchiole dilates to form a condition known as focal emphysema. Gough (1947) and Heppleston (1947, 1954) suggested that this condition develops as a result of weakening and atrophy of the smooth muscle in the bronchiolar wall. The site at which focal emphysema develops is identical to that of the centrilobular emphysema found in cigarette smokers. Some researchers, however, believe that the focal emphysema of coal workers seldom extends to involve the gas-exchanging regions of the lung, namely, the respiratory bronchioles and alveoli (Heppleston 1972). Heppleston (1972) and Gough (1968), moreover, claimed to be able to distinguish focal emphysema from centrilobular emphysema, and suggested that the former is characterized by an absence of bronchiolitis in the smaller airways. Not all researchers accept these opinions (Cockcroft, Seal et al. 1982).

Dust exposure has long been associated with increasing severity of focal emphysema. Gough (1968) wrote that in a young coal miner with short exposure to dust, dying of accident or of nonpulmonary disease, there is an accumulation of coal dust specifically related to the terminal and respiratory bronchioles. The lungs can evidently withstand this deposition without harm for some years. Emphysema then develops, and in miners who have been exposed for 20 years, some degree of dilatation of the proximal order of the respiratory bronchiole is usual and may be marked. After 40 years of dust exposure, the majority of miners will show focal dust emphysema (FDE), although there is a surprising range in the quantity of dust deposited, and in the degree of emphysema, in miners working under similar conditions. FDE refers to dilatation of the respiratory bronchioles and there can be no doubt, because of the time sequence, that the dust deposition precedes the emphysema. Although Gough's remarks imply that there is a direct relationship between dust exposure and the development of focal emphysema, until recently his views were not entirely accepted. In a similar context, there is a clear-cut relationship between coal dust exposure and the development and progression of simple CWP (Jacobsen 1980).

Ryder and colleagues (1970) reported the results of a survey in which they correlated pathological, physiological, and radiological findings from the lungs of 247 deceased South Wales miners and ex-miners, most of whom had been diagnosed as suffering from coal workers' pneumoconiosis during life. The researchers were particu-

larly concerned with the relationship of emphysema to dust exposure and to the radiological findings present antemortem in these subjects. A control series of autopsies was drawn from nonmining men autopsied at the same hospital and matched for age by decade. Whole lung sections were made and emphysema was quantified with standard techniques. Virtually all of the mining population had been examined by the Pneumoconiosis Panel during life, and most were receiving benefits. Post-mortem findings of emphysema were then related to clinical findings, ventilatory capacity measurements, and radiological findings. Emphysema was much more common among the disabled coal miners than among the control population of nonminers, but it is difficult to interpret this observation, as the miners were largely selected from among those who had respiratory disability and the control population was not selected in any similar way. They also found that miners with the punctate type of opacity were more likely to have emphysema than those with nodular or micronodular lesions. Lung function showed no correlation to progressive x-ray changes for simple pneumoconiosis, but declined with increasing severity of progressive massive fibrosis. The mean emphysema score increased with increasing age in the control population, but not in the miners. The absence of a relationship between emphysema score and age in the miners may be secondary to their having been selected (even at the younger ages) because they presented with respiratory disability. The mean emphysema score correlated well with antemortem measurements of FEV_1, but was not greater in those miners with categories B and C of progressive massive fibrosis than in miners with lesser degrees of radiologic change. The absence of smoking data in this population of disabled miners and the poor correlation of emphysema score with radiologic change makes it difficult to ascertain the relative contributions that cigarette smoking and coal dust exposure may have made to the emphysema found in this population.

A later publication on the same population of disabled miners (Lyons et al. 1972) included some smoking data. Lung function declined with increasing severity of radiologic progressive massive fibrosis, but actually improved with increasing severity of radiologic simple pneumoconiosis. This dichotomy of lung function and radiograph may be due to the selection of the autopsy population largely from those who had been disabled from pneumoconiosis in life, as the certification of disability may require more severe functional abnormalities in the absence of radiographic abnormalities than it would in the presence of advanced simple pneumoconiosis on the radiograph. They again showed a correlation of lower FEV_1 with increasing emphysema score, but not with the Reid index of bronchitis. Smokers had lower mean FEV_1 values than nonsmokers and ex-smokers among miners with simple pneumoconiosis and

grade A PMF, but there was no difference in mean FEV_1 for smokers and nonsmokers among workers with more advanced PMF. The authors suggested that the emphysema is a more important determinant of ventilatory impairment than the radiograph and that the emphysema is due to coal dust in both simple pneumoconiosis and progressive massive fibrosis. However, they presented no data to evaluate the possibility that emphysematous change due to cigarette smoking may have been responsible for the link between emphysema score and lung function and for the absence of a correlation with the radiologic changes of pneumoconiosis.

Leigh and colleagues (1983) described the results of 886 post-mortem examinations of Australian miners, relating years spent underground at the coal face to bronchial gland wall ratio, the presence and extent of emphysema in the lungs, radiographic findings, and cigarette smoking history. Emphysema was related to years spent underground at the coal face and to radiological evidence of CWP. Radiological evidence of pneumoconiosis was negatively associated with smoking. Even more surprisingly, smoking was not correlated with gland wall ratio or emphysema. This absence of any relationship between cigarette smoking and emphysema is unique in the published literature and suggests a bias in the selection of subjects who underwent post-mortem examination or in the manner in which smoking habits were analyzed.

A relatively recent post-mortem study of coal miners and nonminers from South Wales compared the prevalence and extent of emphysema in subjects who had died of ischemic heart disease (Cockcroft, Berry et al. 1982). A greater percentage of smokers and ex-smokers had emphysema (17/34) than never smokers (1/5) among the coal miners, but there were too few cases of emphysema among the nonminers to compare smokers and nonsmokers. Coal miners were noted to have more emphysema than nonminers, but the frequency of emphysema in the control population was very low. While the degree of emphysema in these subjects was quantitated in the absence of knowledge of the deceased subject's occupation, the characteristic features of coal miners' lungs (i.e., the formation of macules and the presence of pigment and the accompanying focal emphysema) would invariably indicate the deceased subject's occupation during life. There was a legal requirement for a post-mortem examination for coal miners. Whether the pigment present also highlighted and accentuated the emphysema is unknown.

Ruckley and colleagues (1984) examined the lungs of 460 British coal miners at post-mortem examination for signs of dust-related fibrosis and emphysema. Smoking habits had been determined previously by questionnaire. The prevalence of emphysema was 9 percent in the nonsmoking miners whose lungs showed only circumscribed dust accumulations of which any solid center was less

than 1 mm in size, 33 percent in nonsmoking miners with lungs showing one or more palpable lesions between 1 and 10 mm in size, and 75 percent in nonsmokers with PMF. The corresponding prevalences of emphysema among smokers with similar pathologic findings were 52.7 percent, 70.3 percent, and 85.3 percent, respectively. Ex-smokers generally had intermediate percentages. The percentage of the population with any emphysema increased with the increasing content of dust in the lung, but the percentage of the population with more than one-third of the lung affected showed no increase with increasing concentration of dust in the lung. These data suggest that both smoking and coal dust contribute to emphysema, but that extensive emphysematous change is more closely related to extent of cigarette smoking.

Morgan and colleagues (1971) examined lung volumes in coal miners and showed that both cigarette smoking and increasing simple CWP grade increased the TLC and RV, and the effects appeared to be additive. This suggests that simple coal workers' pneumoconiosis is associated with a slight loss of the elastic recoil. Such an observation is best explained by the presence of so-called focal dust emphysema (FDE).

In summary, there is little doubt that simple CWP and dust exposure may lead to the development of focal dust emphysema. The type of emphysema seen in coal miners is probably still best referred to as focal dust emphysema, since there is some evidence that it does not progress to severe centrilobular emphysema (Ruckley et al. 1984) in the absence of cigarette smoking. Whether a morbid anatomical distinction between the two conditions is possible is not certain.

Studies of right ventricular function in coal miners and ex-miners both during life and at post-mortem examination (Morgan and Seaton 1984) have shown that cor pulmonale or right ventricular hypertrophy do not occur except in cigarette smokers or in miners who have PMF (Fernie et al. 1983).

Dust Exposure, Cigarette Smoking, and Ventilatory Function

In a long-term prospective study of 3,581 miners who worked at the coal face, Rogan and colleagues (1973) showed that dust exposure was inversely related to ventilatory capacity. Lifetime cumulative exposures to coal dust were available. The researchers were able to demonstrate a progressive reduction in ventilatory capacity with increasing exposure to dust. The presence of pneumoconiosis was not associated with an additional decrement of ventilatory capacity beyond that due to cumulative dust exposure, smoking habits, and stature. Smokers showed a more rapid decline in FEV_1 than nonsmokers, but an effect of cumulative dust exposure was apparent in both smoking and nonsmoking miners. Among the nonsmokers,

FEV_1 was generally lower in the most dust-exposed group than in the low exposure group, but the rate of decline per year remained the same from age 30 to age 60 in both exposure groups. The age-related regression coefficients were the same in the heavily and lightly dust-exposed nonsmokers. Subjects with PMF were excluded from the analysis. Among smokers, the rate of decline in FEV_1 with age was greater than for the nonsmokers in each exposure category, but the absolute FEV_1 in smokers at a given age was uniformly lower for the group with high dust exposure than the group with low dust exposure.

Kibelstis and colleagues (1973) were also able to demonstrate a slight effect of dust exposure on the ventilatory capacity of their nonsmoking miners. These investigators divided their population according to whether the men had worked at the face, in transportation, in miscellaneous other jobs, or on the surface. Dust measurements performed for these various jobs and work places had shown a gradient, with the greatest exposure at the coal face and least on the surface (Doyle 1970). Nevertheless, individual cumulative dust exposure measurements were not available for the subjects. When the nonsmoking coal face workers were compared with the nonsmoking surface workers, there was a slight but significant difference in FEV_1. Thus, the coal face workers had an FEV_1 of 98 percent of the predicted value; that of the surface workers was 102.4 percent. The difference in FEV_1 for the smoking and the nonsmoking coal face workers was 6 percent; the difference between the smoking surface workers and the nonsmoking surface workers was 10.5 percent. The effects of cigarette smoking were therefore substantially larger than those of dust exposure. Among the ex-smokers and nonsmokers, there was a significant difference in function between coal face workers and transportation workers and their counterparts who worked on the surface. Among the smokers, no such difference was present, with smoking apparently overwhelming the effects of dust. Airways obstruction was three times more common in the smokers than in the nonsmokers.

An extensive West German study of 6,700 workers employed in coal mines, steel works, cement works, asbestos factories, and other heavy industry related dust exposure to smoking habits and other factors (Deutsche Forschungsgemeinschaft 1978). Each subject underwent a clinical examination, chest radiograph, and spirometry along with measurements of airways resistance and arterial blood gas analyses. The study showed that the most important factors related to the prevalence of bronchitis and airways obstruction were age and smoking habits. Among younger workers, there seemed to be an additive effect of smoking, age, and dust, with the combined effect of all three equaling the sum of their separate effects. Among older

workers, smoking appeared to play a relatively greater role in the production of airways obstruction.

Hankinson and colleagues (1977) characterized the physiological impairments that are associated with the inhalation of coal dust and cigarette smoke. This study relied on flow volume curves as a method of assessing ventilatory capacity, but in addition to the standard spirometric measurements, lung volumes were calculated by a radiological method using posteroanterior and lateral chest films. Since TLC could be calculated, it was possible to express the flow rates, not only as a percentage of vital capacity (VC) but also at absolute lung volumes. Four age- and height-matched groups were selected on the basis of their smoking history and on the presence of bronchitis, that is, cough and sputum. Thus the four groups consisted of smokers with bronchitis, smokers without bronchitis, nonsmokers with bronchitis, and nonsmokers without bronchitis. Flow-volume curves of the four groups are shown in Figures 1 and 2. The differences between the four groups reveal that cigarette smoking effects the flows at all lung volumes. In contrast, nonsmoking bronchitics for the most part showed decreased flows at high lung volumes, although there was some mean reduction in flows at lower lung volumes, indicating that the small airways were not entirely spared. When the flows were expressed at absolute lung volumes, it became evident that smokers had an increased RV and an increased TLC. In contrast, the nonsmoking subjects with and without bronchitis had a normal TLC and RV. The increased TLC suggests a loss of retractive forces in the lung and the presence of subclinical emphysema. Bronchitis in nonsmoking subjects was not associated with an increase in TLC.

A number of longitudinal studies have been carried out with groups of coal miners. Love and Miller (1982) followed 1,677 men from five British collieries for 11 years. Loss of FEV_1 was found to increase with cumulative dust exposure, after allowing for age, smoking, and colliery effect. The investigators classified smoking according to the smoking status that was recorded in all three surveys, that is, as nonsmokers, ex-smokers, or current smokers. Miners who were recorded as smokers at the first survey and as ex-smokers at the second and third surveys were designated as intermittent smokers. According to Love and Miller (1982), the average decrement in FEV_1 from the effects of dust was about one-third of that due to smoking. They further stated that if men left the industry because of ill health and respiratory impairment, the average loss of FEV_1 would have been underestimated. This could apply to men who retired from the workforce because of the effects of dust, cigarette smoking, or both in combination.

There are several problems with this study. First, only 1,677 subjects were studied, 29 percent of the population of 6,191 in the

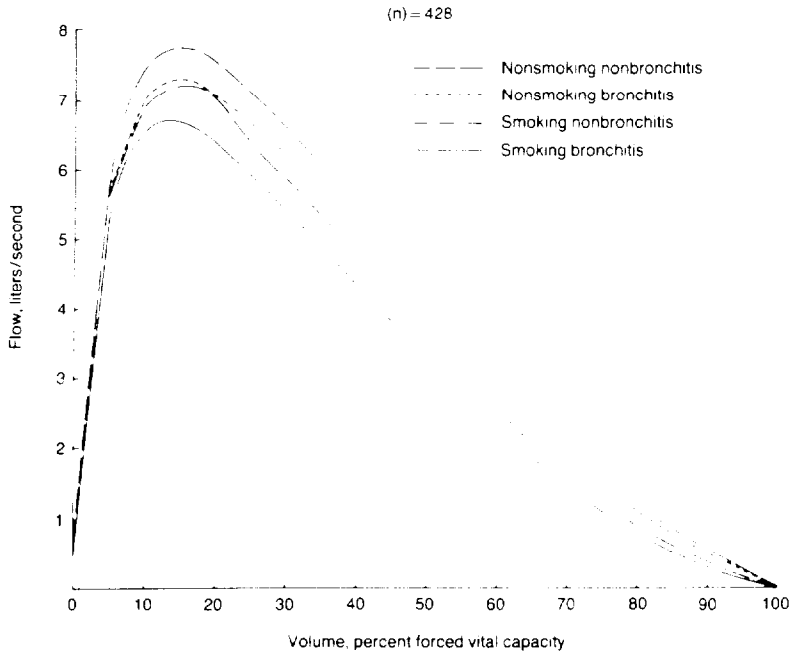

FIGURE 1.—Mean flow volume curves expressed as percentage of forced vital capacity

SOURCE: Hankinson et al. (1977)

first survey. No data were provided to establish that the 1,677 survivors had similar characteristics to the original 6,191 subjects. It is essential to know that the ventilatory capacities, age, height, and smoking habits of the survivors did not differ from those of the original cohort. Second, no quantification of smoking was available. FEV_1 was regressed against age, regular smoking habit, and dust. Unfortunately, smoking was treated as an unchanging variable such as height, although Fletcher and colleagues (1976) had shown that the effects of cigarette smoking increase with pack-years. Similarly, Kibelstis and colleagues (1973) showed that while cough and sputum relate well to current smoking habits, pack-years are a better predictor of the prevalence of airways obstruction.

Attfield (1985) examined changes in ventilatory function among smoking and nonsmoking miners in the United States. They recorded the decline in FEV_1 over an 11-year period in a group of 1,072 U.S. miners. Over that time the loss in FEV_1 was 0.1 L more in

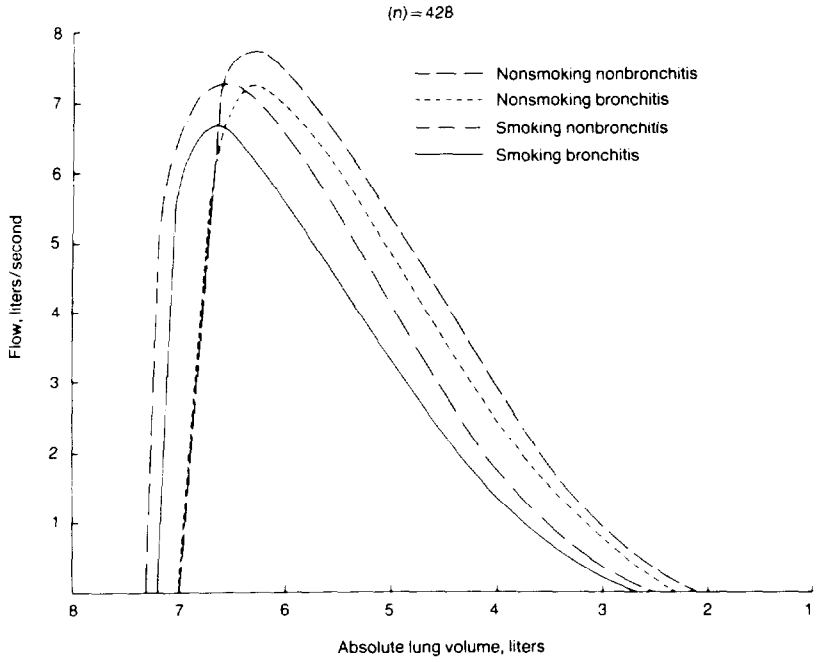

FIGURE 2.—Mean flow volume curves expressed as absolute lung volumes

SOURCE: Hankinson et al. (1977).

the smoking miners than in the nonsmoking miners, in a multiple regression model. The effect of coal dust exposure over the 11-year period ranged from 0.036 to 0.084 L, depending on the regression model used for the coal dust exposure.

The relative importance of cigarette smoking versus dust and other factors in occupational lung disease has been reviewed by Elmes (1981). He concluded that while control of occupational exposure to coal dust remains critical, substantial future improvement in respiratory health can be achieved by reducing the prevalence of smoking among miners.

Summary and Conclusions

1. Coal dust exposure is clearly the major etiologic factor in the production of the radiologic changes of coal workers' pneumoconiosis (CWP). Cigarette smoking probably increases the prevalence of irregular opacities on the chest roentgenograms

of smoking coal miners, but appears to have little effect on the prevalence of small rounded opacities or complicated CWP.

2. Increasing category of simple radiologic CWP is not associated with increasing airflow obstruction, but increasing coal dust exposure is associated with increasing airflow obstruction in both smokers and nonsmokers.
3. Since the introduction of more effective controls to reduce the levels of coal dust exposure at the worksite, cigarette smoking has become the more significant contributor to reported cases of disabling airflow obstruction among coal miners.
4. Cigarette smoking and coal dust exposure appear to have an independent and additive effect on the prevalence of chronic cough and phlegm.
5. Increasing coal dust exposure is associated with a form of emphysema known as focal dust emphysema, but there is no definite evidence that extensive centrilobular emphysema occurs in the absence of cigarette smoking.
6. The majority of studies have shown that coal dust exposure is not associated with an increased risk for lung cancer.
7. Reduction in the levels of coal dust exposure is the only method available to reduce the prevalence of simple or complicated CWP. However, the prevalence of ventilatory disabilities in coal miners could be substantially reduced by reducing the prevalence of cigarette smoking, and efforts aimed at reducing ventilatory disability should include efforts to enhance successful smoking cessation.

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CHAPTER 8

SILICA-EXPOSED WORKERS

CONTENTS

Silica Exposure

The oxides of silicon (SiO_2) are found in a number of polymorphic structures consisting of three-dimensional networks of silica tetrahedra (Zoltai and Stout 1984). When SiO_2 is bound with cations, it is considered to be "combined" silica. When not combined, it exists in its "free" forms—polymorphic crystalline, cryptocrystalline (minute crystals), and amorphous (noncrystalline) (Parkes 1982). Whether SiO_2 is in a free form is important from the standpoint of occupational toxicity. The crystalline phases of SiO_2, including quartz, tridymite, and cristobalite, are recognized as causative agents in silicosis. Diatomite tends to form amorphous silica, and crystalline lenses are found in diatomaceous earth deposits. Diatomite is converted to the biologically active cristobalite with calcining at temperatures from 1000° C to 1723° C (Parkes 1982).

Occupational exposures to free silica are diverse. Major industries with recognized significant silica exposures include metal mining, coal mining, and nonmetallic mineral extraction, stone, clay, and glass processing, iron and steel foundries, and nonferrous foundries. A more complete listing of occupations with potential exposure to silica is found in Table 1. Some of these exposures, including silica flour production and use, sandblasting and certain mining, quarrying and tunneling operations, result in exposure predominantly to silica. However, many silica exposures, including most mining operations and foundry exposures, are mixed-dust exposures, which has implications for the type and extent of biological response seen among exposed workers.

A general pattern of noncompliance with the current permissible exposure limit (PEL) for free silica has been documented in recent papers appearing in the American medical literature. These reports have included the Occupational Safety and Health Administration (OSHA) compliance data for 205 foundries in which 40.6 percent of samples exceeded the PEL (Oudiz et al. 1983); OSHA data showing a 53 percent rate of noncompliance with the silica PEL in 27 silica flour mills (Banks, Morring, Boehlecke 1981; Banks, Morring, Boehlecke et al. 1981); and gross excesses (greater than hundredfold) of the silica standard in sandblasting operations, which remain a poorly regulated industrial process in the United States but are banned in several other developed countries (Samimi et al. 1974).

Population at Risk

Estimates of the population at risk for potential silica exposure are available from the National Institute for Occupational Safety and Health (NIOSH) National Occupational Hazard Survey, which was based on a probability sample of 5,000 industries between 1972 and 1974 (NIOSH 1978). From these survey results, NIOSH estimated

TABLE 1.—Occupations with potential exposure to silica

Abrasive blasters	Oil purifiers
Abrasive makers	Oilstone workers
Auto garage workers	Optical equipment makers
Bisque-kiln workers	Paint mixers
Brick layers	Polishing soap makers
Buffers	Porcelain workers
Burhstone workers	Pottery workers
Carborundum makers	Pouncers (felt hat)
Casting cleaners (foundry)	Pulpstone workers
Cement makers	Quarry workers
Cement mixers	Quartz workers
Ceramic workers	Refractory makers
Chemical glass makers	Road constructors
Chippers	Rock crushers
Coal miners	Rock cutters
Construct workers	Rock drillers
Cosmetic makers	Rock grinders
Cutlery makers	Rock screeners
Diatomaceous earth calciners	Rubber compound mixers
Electronic equipment makers	Sand cutters
Enamellers	Sand pulverizers
Fertilizer makers	Sand blasters
Flint workers	Sandpaper makers
Foundry workers	Sandstone grinders
Furnace liners	Sawyers
Fused quartz workers	Scouring soap workers
Glass makers	Silica brick workers
Glaze mixers (pottery)	Silicon alloy makers
Granite cutters	Silver polishers
Granite workers	Slate workers
Grinding wheel makers	Smelters
Grindstone workers	Sodium silicate makers
Hard rock miners	Spacecraft workers
Insecticide makers	Stone bedrubbers
Insulators	Stone cutters
Jewelers	Stone planners
Jute workers	Street sweepers
Kiln liners	Subway construction workers
Masons	Tile makers
Metal buffers	Toothpaste makers
Metal burnishers	Tube mill liners
Metal polishers	Tumbline barrel workers
Miners	Tunnel construction workers
Mortar mixers	Whetstone workers
Mortarmen	Wood filler workers

SOURCE: Aspen Systems (1978).

that 3,200,000 active workers in 238,000 plants were potentially exposed to silica; however, this estimate was based on workers in an area where free silica is used, and the number of workers with clinically significant exposures would be appreciably lower. This estimate excluded former workers who were retired, working elsewhere, or disabled and the large industrial sector of agriculture where some silica dust exposure occurs (Popendorf et al. 1982).

Significant physical factors for occupational respiratory disease risk include the percentage of free silica, the respirable fraction of the mineral dust (which may have a higher silica content (Ayer et al. 1973)), and the concentration of dust (total and respirable) in the worker's breathing zone. In addition, other workplace contaminants may combine with silica particles to alter the toxicity of the given mineral dust exposure. Individual factors such as pulmonary deposition and clearance, atopic status, genetic constitution, and immune response may also be important risk factors in silica-related disease; however, they are sometimes difficult to measure and have been less well studied. Hence, studies of workers exposed to silica must provide clear documentation of the exposures in the workplace as well as documentation of other personal and environmental factors that may influence biological response.

Smoking Behavior of Silica-Exposed Workers

The smoking behavior of workers in a variety of settings where silica exposure can occur is detailed in Table 2. These studies in the United States and abroad indicate that a very large proportion of people who are exposed to silica are also smokers.

Definitions of Health Effects

Several health effects are associated with occupational exposure to silica dust. The causal role silica plays in some disease responses, namely silicosis and silica-induced alveolar lipoproteinosis ("acute silicosis"), is quite clear and widely accepted. Silica is recognized as playing an important causal contributing role in a second group of pulmonary responses—silicotuberculosis, mixed-dust fibrosis (usually mixed with iron oxides), and the fibrosing alveolitis arising from exposure to calcined diatomaceous earth (diatomite pneumoconiosis) (Parkes 1982). Smoking appears to play no significant causal role in the etiology of the first two categories of silica-induced diseases. In a third group of health effects, silica dust appears to be a risk factor in simple chronic bronchitis as characterized by mucus hypersecretion and in chronic airways obstruction, which is often associated with a progressive decline in expiratory flow rates and is largely irreversible. This last group of pulmonary responses is nonspecific, recognized to be multifactorial and causally linked to cigarette smoking. A causal relationship between silica dust and chronic bronchitis or chronic airways obstruction is less clear. This issue is of considerable importance because of the prevalence of chronic bronchitis and chronic airways obstruction in modern society and the large size of the population at potential risk of silica exposure.